FACTS

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ABOUT PHYTOTOXICOLOGY

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HOW AIR POLLUTION AFFECTS VEGETATION

Responses to air pollution can become manifest in various ways. Pollutants can injure vegetation, endanger human and animal health, soil buildings and clothing, contribute to highway accidents by reducing visibility, help depress property values and generally interfere with our aesthetic enjoyment of the landscape.

Vegetation injury due to air pollution is an area of particularly serious concern. It can range from visible markings on foliage to reduced growth and yield to premature death of plant life. The ensuing visual and economic consequences can at times be disastrous. Injury to crops possessing marketable foliage such as lettuce or tobacco can result in especially high losses.

Investigation of Vegetation Damage in Ontario

The Air Resources Branch of the Ministry of the Environment is responsible for the assessment of air quality and its effects to aid in the control and prevention of air pollution in Ontario.

The Phytotoxicolgy Section is responsible for determining the degree and extent of air pollution injury to all types of vegetation throughout Ontario. (Any pollutant that injures vegetation is a phytotoxicant.) The section pursues its objectives by:

1. Investigating requests from the public concerning suspected air pollution injury to vegetation -- forests, orchards, farm crops, ornamental plantings -- in both rural and urban areas. In so doing it is necessary to differentiate pollution injury from similar injuries caused by insects, disease, adverse weather, poor nutrition or mismanagement.

2. Conducting assessment studies in areas of concern where adverse effects on vegetation may occur as a result of emissions from existing or future sources of air pollution. If ambient air quality records coupled with vegetation data indicate the biological component of the environment to be in danger, then prompt abatement action is taken.

3. Carrying out practical research studies under controlled environment conditions on the effects of air pollutants on vegetation. These studies are conducted to complement field investigations, screen resistant plant species and determine air quality criteria for the protection of agriculture and forestry.

The staff of the Phytotoxicology Section consists of forest and plant pathologists, agricultural specialists, plant ecologists, soil specialists, a histopathologist, a biostatistician, and greenhouse and laboratory technicians. The Phytotoxicology controlled environment facility is located in Brampton.

In the Phytotoxicology laboratory, vegetation samples collected during complaint or assessment visits are examined by pathological and histological techniques, and processed for chemical analysis. A herbarium is maintained to demonstrate, compare, and diagnose plant material damaged by particular air pollutants.

Studies conducted in some areas include the growing of plants in specially designed shelters equipped or not equipped with devices to filter the ambient air. Certain plant species and varieties which are especially sensitive to various air pollutants are raised in a filtered-air greenhouse under uniform culture for use in field experiments.

The Environmental Protection Act, 1971, has made provision for a Board of Negotiation to mediate the settlement of claims of persons whose forests, crops, or livestock are damaged by air pollution and have suffered an economic loss.

Phytotoxicology personnel investigate over 200 air pollution vegetation complaints each year. Vegetation suspected of being injured by air pollutants included ornamental flowers, shrubs and trees; garden fruits and vegetables; stored vegetables; greenhouse flowers and crops; farm crops (white beans, tomatoes, green onions, winter wheat, oats and corn), animal pastures and cured hay; and fruit and forest trees.

Suspected air pollutants and those ascertained as causing vegetation injury included fluorides, sulphur dioxide, ozone, peroxyacetyl nitrate, acid rain, boron, lead, chlorine, hydrogen chloride, arsenic, zinc, chromium, nickel, cobalt, salt spray, urea, nitrogen dioxide, ammonia, cement dust, magnesium-lime dust, and soot.

The Phytotoxicology Section maintains a close surveillance of vegetation in areas of concern throughout Ontario. Baseline studies are conducted in agricultural or forested areas before a major pollution source becomes operational to determine the pre-operational endemic conditions. Ecological studies keep the Section informed of increasing or decreasing vegetation effects in the vicinity of pollution sources.

Over 3,000 assessment station visits to areas of concern are made annually by Phytotoxicology personnel. During both public request and assessment investigations, over 10,000 soil and vegetation samples are collected each year for laboratory examination.

Effects of Air Pollutants on Plants

Air pollution injury to plants can be evident in various ways. Injury to foliage may become visible in a short time and take the form of necrotic lesions (dead tissue) or it can develop slowly and become manifest as a yellowing or chlorosis of the leaf. There may be a reduction in growth of various portions of a plant. Plants may be killed outright but they usually do not succumb until they have suffered injury perennially.

Injury may not be visible externally with effects occurring sub-cellularly in cell membranes and chloroplasts (plant organelles where photosynthesis takes place). The plants may suffer physiologically due to an upset in the rate of photosynthesis, respiration or transpiration.

Sulphur Dioxide

There is reference to the deleterious effects of sulphur dioxide on vegetation dating back more than 100 years in Europe. In the United States the Experiment Station of the Agricultural College of Utah published a bulletin in 1903 describing the effects of smelter smoke on Utah agriculture. In the 1930's an international problem arose when smelter fumes emitted by the Consolidated Mining and Smelting Company at Trail, British Columbia travelled down the Columbia River Valley to damage forests in Stevens County in the State of Washington. Comprehensive investigations were carried out for about 10 years resulting in the publication of a book by the National Research Council of Canada in 1939. Investigations in the Sudbury district of Ontario started in the 1940's and are still continuing.

Major sources of sulphur dioxide are coal burning operations, especially those providing electric power and space heating. Large quantities of sulphur dioxide can also result from the burning of petroleum fuels and the smelting of sulphur-containing ores.

Sulphur dioxide enters leaves mainly through the stomata (microscopic openings where normal gas exchanges of oxygen and carbon dioxide occur). The toxicity of sulphur dioxide to the mesophyll cells (inner chloroplast-containing cells) of leaves is primarily due to its reducing properties.

Leaf injury is classified as either acute or chronic. Acute injury is caused by absorption of high concentrations of sulphur dioxide in a relatively short time. This results in a rapid accumulation of sulphite which is toxic to the metabolic processes taking place in the mesophyll cells.

Chronic injury is caused by long-term absorption of sulphur dioxide at sub-lethal concentrations. The sulphite formed is oxidized to sulphate at about the same rate that the gas is absorbed. When sulphate accumulates beyond a threshold value that the plant cells can tolerate, chronic injury occurs. It is estimated that sulphate is about 30 times less toxic than sulphite. Chronic effects on trees include foliar discoloration, premature shedding of leaves, reduced radical growth and early death of the trees.

Acute sulphur dioxide injury on broad leaves takes the form of bifacial lesions, which usually occur between veins, and is often more prominent towards the petiole (leaf-stalk). The injury is local. The metabolic processes are completely disrupted in the necrotic or dead areas, with the surrounding leaf tissue remaining green and functional. The green pigments are decomposed and the affected leaf area assumes a bleached, ivory, tan, orange-red, reddish-brown or brown appearance, depending upon the plant species, time of year and weather conditions. The tissue on either side of the veins is extremely resistant. In some cases, injury can occur on the margins of leaves.

Young leaves rarely display necrotic markings whereas newly expanded leaves are most sensitive to sulphur-dioxide injury. The oldest leaves are moderately sensitive. In monocotyledonous (blade-like) leaves the injury can occur at the tips and in lengthwise areas between the main veins. In conifers acute injury usually appears as a bright orange-red tip necrosis on current-year needles, often with a sharp line of demarcation between the injured tips and the normally green bases. Occasionally the injury may occur in bands, in apical, medial or basal locations on the needles.

Chronic sulphur-dioxide injury becomes manifest as a yellowing or chlorosis of the leaf, sometimes from lower to upper surfaces on broad leaves. Occasionally only a bronzing or silvering will occur on the undersurface of the leaves. The rate of metabolism is reduced in leaves displaying chronic injury. In conifers chronic injury on older needles is first indicated by [illegible]

Different plant species and varieties and even individuals of the same species may vary considerably in their sensitivity or tolerance to sulphur dioxide. Susceptibility lists have been made by several investigators but they can be only used as a guide. Variations can occur because of differences in geographical location, climate, and plant stage of growth and maturation.

Vegetation sensitive to sulphur dioxide include alfalfa, barley, eastern white pine, white birch, white ash, trembling aspen, Chinese elm, Manitoba maple and bracken fern.

In cities, trees found resistant to sulphur dioxide pollution in descending order are Ailanthus, pin oak, gingko, Carolina poplar, London plane, Norway maple and little-leaf linden.

Environmental factors conducive to optimum plant growth usually abet sulphur-dioxide injury. They include sunlight, moderate temperature, high relative humidity, wind and adequate soil moisture.

Most investigators have shown a direct relationship between open stomata and the absorption of sulphur dioxide and subsequent leaf injury. When stomata are closed, either at night because of darkness or during the day because of other factors, plants are more resistant to sulphur dioxide. It has been reported that the potato plant is as equally sensitive at night or during the day because their stomata do not close at night.

Vegetation is most susceptible to sulphur dioxide during the active growth months of June, July and August. For acute foliar injury to occur, 0.25 parts per million (ppm) of sulphur dioxide for eight hours or 0.95 ppm for one hour usually must be present. If the environmental factors and growth stages of the plants are not conducive to injury, the plants will escape injury even in the presence of potentially damaging concentrations of sulphur dioxide. Chronic effects have been documented on forest trees exposed to an annual average of about 0.02 ppm sulphur dioxide.

Fluorides

Fluoride injury to vegetation was recognized in Germany over 70 years ago. In addition to vegetation damage, livestock was affected in the vicinity of certain industries.

Fluorides may be discharged into the atmosphere from the combustion of coal; the production of brick, tile, enamel frit ceramics, and glass; the manufacture of aluminum and steel; and the production of hydrofluoric acid, phosphate chemicals and fertilizers.

Sensitive vegetation may be injured when exposed for 24 hours to atmospheric concentrations of hydrogen fluoride of 1 part per billion (ppb). Similar injury symptoms may be produced by higher concentrations for shorter periods of time. The amount of fluoride accumulated in plant tissues depends on the absorption capacity of the plant, its sensitivity to fluorine and ambient air concentrations. High concentrations of fluoride may accumulate in leaves during the growing season while subjected to extremely low concentrations in the air. Bitternut hickory can concentrate up to 1,000 ppm fluoride in its leaves without showing any visible injury, whereas the sensitive gladiolus may exhibit leaf injury with less than 35 ppm fluoride.

Fluorides absorbed by leaves are translocated towards the margins of broad leaves and to the tips of monocotyledonous leaves and coniferous needles. Little injury takes place at the sites of absorption, whereas the margins or tips of the leaves build up

Fluoride injury starts as a gray or light-green water-soaked lesion which turns tan to reddish-brown. It can appear within a few hours of a week after exposure depending on plant species and variety, the concentration of atmospheric fluorides, the duration of exposure and various environmental conditions. With continued exposure, the necrotic areas increase in size spreading inward to the mid-rib on broad leaves and downward on coniferous needles.

Fluorides inhibit photosynthesis, the impairment being measurable even before visible leaf injury occurs. With continued fumigation, the decrease in photosynthesis rate parallels the increase in leaf tissue necrosis. Fluorides inhibit enzymes in vitro. A well-known example is enolase, an enzyme required in the glycolytic pathway of plant respiration.

Studies of plant species susceptibility to fluorides showed that pine (developing needles), gladiolus, apricot, prune, plum, grape, tulip, iris, St. Johnwort and sweet corn were most sensitive.

Atmospheric fluorides, by concentrating in foliage and directly injuring plants, pose a threat to the health of livestock. Forage crops may appear normal while actually containing high concentrations of fluroide. Alfalfa, for example, can tolerate several hundred ppm fluoride without showing visible injury. Cattle, feeding on this plant over an extended period of time, may develop the disease fluorosis if the fluoride content is in excess of 40 ppm. The symptoms of chronic fluorine toxicosis are mottled and abraded teeth, swollen periosteal (bone surface) tissue, lameness and, in severe cases, decreased appetite and milk production.

Ozone and PAN

Ozone and PAN (peroxyacetyl nitrate) are the main phytotoxicants in the Los Angeles type of oxidant smog now plaguing many urban areas. Automobile exhaust is the major contributor of the primary pollutants (nitrogen oxides and reactive hydrocarbons) in the photochemical reaction producing the secondary toxic pollutants (ozone and PAN).

Oxidant damage to plants was first observed in the Los Angeles area in 1944. A wide variety of plants are susceptible to oxidant damage. In southwestern Ontario, phytotoxicology surveys conducted annually have revealed the widespread occurrence of ozone injury on tobacco, tomato, potato and white bean crops. Ozone causes a spotting, bleaching or chlorosis of upper leaf surfaces. Typical lesions are produced on tobacco plants by concentration of ozone as low as 0.05 ppm for four hours, crop yields are reduced if exposed to an average of 0.04 ppm of ozone over the growing season during daylight hours. PAN causes bronzing, silvering, or glazing of lower leaf surfaces. Sensitive plants, such as tomato and lettuce have been injured by 15 to 20 ppb of PAN in a four hour exposure. Light is necessary before, during and after a fumigation by PAN to cause visible injury.

Susceptibility to ozone injury is influenced by environmental and plant factors. It is increased by high relative humidity and low carbohydrate content. Ozone injury to broad leaves displays a definite pattern related to the development of functional stomata. The youngest leaves are resistant and with expansion become susceptible at their tips. With increasing maturity the leaves become successively susceptible at middle and basal portions. The leaves become resistant again at complete maturation. Peculiarly, ozone usually enters through the stomata on lower leaf surfaces but injures palisade mesophyll cells in the upper layers of the leaf. In these cells the chloroplasts disintegrate followed by plamolysis (contraction) and desiccation (drying up) of the cellular contents.

In addition to visible injury, growth suppression may result from the effects of

Experimental work has shown that sulphur dioxide and ozone may act synergistically to reduce the required concentrations of either gas to produce leaf injury. Ozone as low as 0.027 ppm when combined with 0.24 ppm sulphur dioxide injured Bel W3 tobacco plants in two hours.

Nitrogen Dioxide

Nitrogen dioxide (NO_2) in high concentrations can cause plant injury symptoms similar to those caused by sulphur dioxide. Under high light intensities, about 6.0 ppm of NO_2 for two hours is required to injure sensitive plant species, such as pinto bean, tomato, and cucumber. Low light intensity increases sensitivity of plants with injury developing after exposure to 2.5 - 3.0 ppm of NO_2 for two hours. Nitrogen dioxide can injure the same plants as ozone and in the same physiological tissue. Injury symptons are different, however, and approximately 10 times as much nitrogen dioxide is required. Long-term exposures of tomato plants to low concentrations of nitrogen dioxide (up to 0.5 ppm for 10 to 22 days) may inhibit plant growth, and increase the green color (chlorophyll content) of the leaves. Experimental work has shown that low levels of NO_2 (0.10 ppm) in combination with SO_2 (0.10 ppm) acted synergistically injuring a number of plant species in a 4 hour exposure period.

Chlorine

Chlorine is not widespread in the atmosphere. It is usually confined to the immediate area surrounding its source. High concentrations of chlorine released in tank car accidents can cause severe defoliation and leaf injury to contiguous vegetation. The symptoms of chlorine injury are quite diverse, and range from terminal and marginal necrosis and chlorosis, to interveinal lesions occuring both bifacially and on upper leaf surfaces only.

Alfalfa and radish plants are injured by 0.1 ppm of chlorine for two hours. This threshold dosage places chlorine between fluorides and sulphur dioxide in phytotoxicity.

Particulate Matter

Particulate matter such as cement dust, magnesium-lime dust and carbon soot deposited on vegetation inhibit photosynthesis. Cement dust may cause chlorosis and death of leaf tissue by the combination of a thick crust and alkaline toxicity produced in wet weather. Deciduous and coniferous trees are injured, the latter occasionally killed. Accumulation of alkaline dusts in soil can increase soil pH to levels adverse to crop growth. Sulphuric acid aerosols produce punctate spots on the upper surfaces of wet leaves.

De-icing compounds applied to roads and highways in winter can result in injury to roadside vegetation caused by splashing of the salt by passing vehicles. Fruit trees such as peach and apple are particularly susceptible to salt spray. The severity of twig dieback injury on the trees decreases with distance from the road and becomes negligible usually beyond 150 feet.

Investigating Air Pollution Injury to Vegetation

Any resident of Ontario who suspects that plant life (ornamentals, crops, orchards or woodlands) or soil in urban or rural areas is being affected by air pollution can request an investigation by the Phytotoxicolgy Section. If the injury or damage is diagnosed as being caused by air pollution and the source is detected, a report of the investigation is sent to both the complainant and the offending source. Abatement engineers are notified to inspect the offending source to prevent further phytotoxic emissions.

The Air Resources Branch encourages private settlement of damages. If this is not feasible, the claimant can request mediation by the three-man Board of Negotiation.

The investigation of an air pollution vegetation complaint at times may be compared to a "Whodunit". Research experience and the application of detective work to plant pathology techniques are essential. Some complaints are solved readily; others require lengthy investigations.

One complainant property in a northern Ontario study was surrounded by seven industries. The local abatement engineer wanted to know what pollutant caused the vegetation injury and which industry was responsible. Through careful investigation of the pattern of injury on both complainant and neighbouring properties, through the knowledge of air pollution injury symptoms and the susceptibility and resistance of various plant species, by examining wind records and by chemically analyzing collected vegetation, it was found that the injury was caused by an acute fumigation of sulphur dioxide and that the source was a sulphite pulp and paper mill. Not only were the complainant and the offending source brought together for settlement of damages, but the industry also purchased new control devices to prevent any further accidental release of high concentrations of sulphur dioxide.

In another investigation of an acute episode, the vegetation injury area was situated between two industries -- an aluminum chloride manufacturer about one mile west and a nickel refinery about one mile southwest. Phytotoxicology personnel made a detailed vegetation survey in the area. No pollution injury was observed near the industry to the west or at locations midway between the injury area and this industry. In the injury area, it was found that trees, shrubs and hedges displayed severe foliar injuries only on their southwest sides. Samples of collected foliage and soil were analyzed for several pollutants that the two industries could emit, such as sulphur, fluoride, chloride, aluminum, copper and nickel. The results showed that sulphur and fluoride contents were normal when compared to control collection analyses, that chloride, aluminum, and copper levels were slightly elevated, whereas nickel contents were present in excessive and toxic quantities. Since nickel was one of the pollutants emitted from the industry to the southwest, this industry was implicated as the offending source.

About 50 per cent of the injuries investigated by the Phytotoxicology Section are found to be due to causal agents other than air pollutants. For example, several complaints were received by the Phytotoxicology Section concerning injury to European mountain ash trees in the City of Cornwall. Upon investigation, it was found that some of the injuries had indeed been due to air pollutants from local sources, but that for the most part the injuries were caused by the disease Cytospora canker which was rampant in the area at the time.

In North Bay, a number of complaints were received regarding heavy particulate fallout assumed to be originating from the superstack at Sudbury 80 miles away. The phytotoxicology investigation showed that the particulate matter was organic in nature and occurred only near white birch trees. The particulate matter was diagnosed as droppings excreted by an insect, the birch skeletonizer, which was heavily infesting white birch trees throughout northern Ontario at that time.

In Sarnia, residents complained in the spring that their houses had been splattered by "purple rain". Samples of the purple spots on paper and some debris collected from the area were examined in the Phytotoxicology laboratory. The spots were found to be caused by a purple pigment which had leached out of specks in the debris, and under the microscope the specks were identified as anthers from white elm flowers. Apparently a rainstorm had dislodged the anthers from the flowers on white elm trees and splattered them on the sides of houses causing the purple spots.

In early June of a recent growing season several complaints were received from the Port Maitland area that maple trees had suddenly developed leaf injuries on the sides of the trees facing a sulphuric acid making plant. A comprehensive investigation was carried out and it was found that this particular leaf scorch was widespread throughout the Niagara peninsula area of Southern Ontario. Chemical analyses, air sampling records, and other studies showed that air pollutants were not responsible for the injuries. Only succulent foliage on silver maple, sugar maple, red maple, and beech trees were found to display the injury symptoms which appeared suddenly in response to weather changes in which a wet, cloudy period was followed suddenly by a sunny, windy period. This abnormality has been named Late-Spring Leaf Scorch (LSLS).

These cases illustrate how other casual agents can injure vegetation creating symptoms that mimic those brought on by air pollution. When it is determined that injury is being caused by a biological agent or by poor management, the grower is advised to seek combative measures or management advice from the agency authorized to handle such matters.

Prevention of Phytotoxic Effects

The protection of plants from the adverse effects of aerial phytotoxicants cannot be carried out in exactly the same manner as is possible with disease-causing, organic reproductive bodies. A pollution-diseased plant cannot infect another plant; thus there is no need for a quarantine or for eradication of the affected plants. In certain instances, sprays and dusts have protected plants from air pollution injury. The development of resistant varieties holds some promise. The best control method, however, is to reduce the concentrations of noxious pollutants at their sources so as not to exceed the established air quality criteria for agriculture and forestry.

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